situation is, what is wrong and what is being done about it. This might easily be conceivable in a complex situation where a number of physicians are involved and no one of them has really communicated with the patient. This could be an understandable and valid reason for a patient to want to examine the medical record. A second and more likely reason might be a kind of morbid curiosity on the part of a patient. There are such patients, and they are the ones likely to misunderstand or misinterpret what they read, often looking things up in a library, and they are prone to develop additional and unnecessary anxieties as a result—and may begin to treat themselves unbeknownst to the physician who is attending them. Or a patient may be trying to check up on the doctor and pass judgment on the treatment. Here the question becomes to what extent is it in the interest of a patient to act as his or her own physician. It is said, and with some reason, that a physician who acts as his own doctor has a fool for a doctor. It is hard to conceive that this role is likely to be in the best interest of a relatively uninformed patient. And yet another, and in these days even more likely, reason for a patient to wish to peruse his medical record is dissatisfaction with the physician which may lead to intent to sue. This is a quite recent phenomenon and has introduced a new purpose for the physician's records. It is now an instrument for the protection of the physician as well as his instrument to help him serve patients.

If things continue as they are it is likely that we shall have "open season" on medical records. The confidentiality of the hospital record has already been pretty thoroughly eroded and a physician's personal office records can now be subpoenaed. It is likely that this is only the beginning. As the trend continues physicians will understandably take greater protective measures and develop alternative means of maintaining the information they need to properly care for their patients and the medical record will be maintained strictly "for the record," including for purposes of the physician's legal defense. Privileged information will be communicated by telephone or other means much as is now done in other situations where consumers have access to records that were previously considered confidential. All of this will add yet another increment to the cost of medical care, and like so many of the new legal and regulatory requirements now imposed upon medicine and the health care system, the added cost will add little if anything to patient satisfaction or to better care. The extra cost for a "benefit" which may actually be harmful to many will be passed on to patients and third party payors, including government. It seems unlikely that much of anything will be gained by anyone, yet this no doubt is what will be done.

—MSMW

Lead Is Where You Find It

TO PARAPHRASE A SONG, "Lead is where you find it/Don't be blinded/It's all around you/Everywhere." Everywhere, indeed; even in Los Angeles. And do not be blinded by an inability to define its threshold of toxicity. The article by Wesolowski and associates in this issue illustrates these two important points—the locale of the lead problem among children and the level of blood lead that is unacceptable are different from what we thought them to be 15 years ago.

Symptomatic childhood lead poisoning in the United States was generally viewed as a health problem limited to the urban, northeastern cities with their blighted housing, although midwestern cities contributed a fair share to the literature on lead poisoning. Beginning in the early 1970's screening programs were conducted in rural areas and small towns. Elevated blood lead levels were found in children from these presumed pristine sanctuaries. In 1973 the Center for Disease Control initiated a Childhood Lead Based Paint Poisoning Prevention Program that funded projects for lead screening of children in all regions of the United States. The California project was one among 77 conducted in 1975, during which time 440,650 children were screened.[1] Children with elevated lead levels were found in communities from Augusta, Maine, to Los Angeles. Nationally, 6.5 percent of all the children tested had elevated blood lead levels confirmed by repeat testing. The data represented populations of young children, selected for their residence in older, deteriorating housing, as was the case in the California project.

Criteria for screening based on the age and condition of housing are reasonable because the major source of lead in symptomatic lead poison-

ing has been peeling, lead-laden paint. Pica was the modus operandi. Other known sources leading to lead encephalopathy have been burning lead batteries and fruit juice stored in improperly glazed earthenware containers. As the interest in lead poisoning expanded from diagnosis and treatment of children with symptoms to identifying asymptomatic children with elevated blood lead levels, other exposures to lead have been recognized. The fallout from automobile exhaust and industrial pollution produces lead-laden dust in homes and schools.[2] Lead smelters fill the surrounding atmosphere with high concentrations of lead. Even efforts to remove lead from walls and woodwork may inadvertently produce lead-laden fumes or spread pulverized fragments that are difficult to remove. Pica, then, is not necessary for increased ingestion of lead. Breathing the contaminated air near a smelter or innocent hand-to-mouth activity with hands coated with contaminated dirt and dust becomes a means of increasing a child's lead intake.[3]

Lead poisoning was initially approached as a clinical entity. Patients had the exposure and symptoms, and the diagnosis was confirmed by anemia, radiographic appearance of the long bones or coproporphyrinuria. The determination of a blood lead level was not often carried out. In the 1960's atomic absorption spectroscopy facilitated the measurement of lead on large numbers of blood samples. The improved technology plus the increased interest in detecting lead poisoning before symptoms developed led to large scale screening programs. The first was conducted in Chicago in 1966. Screening programs depended on the blood lead level as the essential criterion for diagnosis and management. For the Chicago program the cutoff value was 50 μg per 100 ml whole blood. Four years later the Surgeon General of the United States recommended that 40 μg per 100 ml be used as evidence of undue absorption of lead. The upper limit of normal for blood lead according to the Childhood Lead Poisoning Prevention Program is 29 μg per 100 ml.

For years children with blood lead levels in the 30 to 50 μg per 100 ml range were largely ignored. They make up the largest percentage of children who are detected by screening programs. What happened to those children whose lead levels were not high enough to warrant careful surveillance when our standards, ironically, were "too high"? What should be our response now?

The consequences of slight elevations of blood lead levels in children are difficult to ascertain and the difficulty has led to the lexicon used in describing the lead problem. There is *undue absorption of lead*—the statement rightly implying that some absorption is everyone's due. An *increased body burden of lead* is not as bad as *classical lead poisoning*. The terms *subclinical* and *asymptomatic lead poisoning* lead one to question what is actually meant by poisoning. The confusion in establishing criteria and the resort to ambivalent lexicology are due, in part, to careless mixing of indices of absorption or exposure and indices of toxicity. It is well to keep in mind what the various tests mean.

The concentration of lead in the blood is an index of absorption. Repeat testing may show variation between the two samples and in the California project 40 percent of second samplings had a lower lead concentration. Technologic factors may be partly responsible, but it is also important to realize that a blood lead level reflects a single point in the complex kinetics of exchange of lead between red cells and tissue.[4] One can interpret the blood lead level in two ways.

First, it can be used solely as evidence of increased absorption of lead, after one has established the upper limit of normal. In so doing, no effort is made to define what the toxic level is. Therefore, the data of Wesolowski and co-workers indicate that less than half of the children tested had a lead level commensurate with normal exposure (the percentage might have been greater if repeat sampling had been done in all cases).

Another interpretation of the blood lead level relates the value to some index of toxicity, realizing that the lead level itself is not a measure of toxicity. This is far more difficult because the correlation between toxicity and lead level is not always precise and, furthermore, the critical index of toxicity is very difficult to establish.

The sensitivity of the heme synthetic pathway has made measurement of its enzymes and substrates a means of assessing metabolic toxicity. Currently, erythrocyte protoporphyrin (EP) is the most frequently measured metabolite in evaluating children for lead poisoning. Elevation of EP is also a useful test for iron deficiency in children. It is not surprising that in screening children at risk for both lead poisoning and iron deficiency, there will be instances of normal lead level and elevated EP. Such a discrepancy could also occur if sampling were done at the time of an ebb in the fluctuation of blood lead. The EP level is not subject to

wide daily variations, since the concentration within the red cell is constant for the cell's 120-day life span. Although the Center for Disease Control guidelines considered an EP value of less than 60 μg per 100 ml of whole blood as normal, studies indicate that the upper limit of normal in children is closer to 45 μg per 100 ml of whole blood.[5] So the group of children with blood lead levels between 30 and 50 μg per 100 ml and EP values less than 60 μg per 100 ml should not be ignored.

In general, projects such as the one in California, will find that most of the children with elevated lead levels and EP values are asymptomatic and the elevations are not pronounced. How to relate the blood level or EP value to the risk of clinical toxicity is the dilemma of assigning a threshold of toxicity. Certainly it is the neuron, rather than the red cell precursor with deranged heme synthesis, that is the chief concern. The manifestation of neurologic damage in asymptomatic children will not be apparent for some years after the period of increased ingestion and is likely to consist of learning or behavioral disturbances. In the most relevant, though not a perfect study, de la Burde and co-workers found that poor academic performance, primarily due to behavioral problems, was more frequent among children with increased exposure to lead during early childhood.[6]

The lead problem in children is a frustrating one. The full impact is not known. Safe elevations cannot be confidently given. Successful management depends on socioeconomic and cultural factors as well as medical practice. Each community must consider whether there is a population of children at risk. Then a screening program must find them. The legacy of old, substandard housing and the likelihood of continued pollution will make lead a problem for a long time.

HAROLD S. ZARKOWSKY, MD
Associate Professor of Pediatrics
Washington University School of Medicine
St. Louis Children's Hospital

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PSRO — Update 1977

THIS IS THE FOURTH in a series of more-or-less annual editorial reports on Professional Standards Review Organization (PSRO) developments. One senses that during the past year slow but steady progress has been made toward implementing the complex and potentially very important PSRO law. It is expected that by the end of this federal fiscal year (30 September 1977) there will be 119 conditional PSRO's in place and 70 planning PSRO's proceeding toward conditional status, with only a few of the designated areas remaining unfunded. In a very few areas no PSRO has yet developed. The present law has extended the time professional organizations will have priority in establishing PSRO's until 1 January 1978. The hope has been expressed by the government that it will not be necessary to invoke the alternative means provided by the law to create PSRO's in those areas which have not been able to establish one.

Much more remains to be resolved at the federal level. The tension between the law's dual priorities of cost control and quality assurance continues, with neither having gained a clear ascendency within the federal bureaucracy. This is probably as it ought to be. Too much emphasis on one adversely affects the other. Attempts are underway at the bureaucratic and legislative levels to clarify the relationships of PSRO's to the state review mechanisms for Medicaid programs and to the federally mandated medical review boards of the national End Stage Renal Disease (ESRD) program. The confidentiality issue also has not been settled. The Privacy Protection Study Commission, created by Congress in 1974, has called upon the Department of Health, Education, and Welfare to require hospitals to adopt procedures to guard the privacy of medical records as a condition of qualifying for Medicare and Medicaid reimbursement. But what actually will be done to protect the privacy of physicians and their patients remains to be seen. Meanwhile there is talk of developing patient, physician and institutional profiles for analysis to make comparisons